

Clinical Radiology Extra (2003) **58**: 29–31
doi:10.1016/S1477-6804(03)00002-5, available online at www.sciencedirect.com

Case Report

Exogenous lipid pneumonia presenting as haemothorax

K.E. PAISLEY*, R.G. BERRISFORD†, S.R. HARRIES*

**Department of Clinical Radiology and †Department of Surgery, Royal Devon and Exeter NHS Trust, Exeter, Devon UK*

Received: 2 October 2002 Accepted: 29 November 2002

INTRODUCTION

Exogenous lipid pneumonia is an uncommon condition, first described in 1925 [1], resulting from inhalation or aspiration of animal, vegetable or mineral oil into the lung. Often, but not always, patients have predisposing risk factors such as neuromuscular and oesophageal abnormalities. Diagnosis is often delayed or even missed as symptoms are frequently absent [2,3] or non-specific and radiological findings can mimic other processes such as lung cancer [4]. Aspiration can occur imperceptibly and histologically proven lipid pneumonia can occur without remembered exposure.

We report a case of exogenous lipid pneumonia in a man with cerebral palsy with distinctive computed tomography (CT) findings but an unusual clinical presentation.

CASE REPORT

A 30 year-old man with cerebral palsy was admitted having been unwell for 4 weeks with non-productive cough, wheeze and dyspnoea not responsive to oral antibiotics. There was no chest pain, haemoptysis or weight loss. Past history included two prior admissions with pneumonia at another hospital, 7 and 11 years previously; the earlier admission was known to have involved the right upper lobe. There was also a longstanding history of abnormal swallowing documented by videofluoroscopy which showed some risk of aspiration of fluids exacerbated by occasional neck extension. Other past history included epilepsy and megacolon that was associated with chronic constipation. Current drug treatment comprised sodium valproate, carbamazepine and senna, although he had been taking liquid paraffin for constipation until several years previously. On examination he was afebrile and tachypnoeic with an oxygen saturation on air of 96%. There was a thoracic scoliosis and dullness to percussion

with reduced breath sounds at the left base and left midzone. Haemoglobin was 12.3 g/dl, white cell count $5.5 \times 10^9/l$ and C-reactive protein 66 mg/l.

Chest radiograph (Fig. 1) showed marked scoliosis and almost complete opacification of the left hemi-thorax. There was also shadowing centrally in the right lung which had not changed since the last film of 5 years previously. Subsequent ultrasound confirmed a large left pleural effusion associated with complete atelectasis of the left lung. An intercostal drain was inserted which drained 2 l of old blood. Analysis of this fluid showed no malignant cells, no excess of white cells, scanty macrophages and mesothelial cells; Gram stain, Ziehl-Nielsen stain and culture were negative. Rigid bronchoscopy did not show any macroscopic endobronchial lesion.

CT was performed using a Siemens Volume Zoom multislice scanner (Siemens, Erlangen, Germany). Scans were obtained supine after intravenous injection of contrast medium (100 ml Niopam 300 at 3 ml/s) and were reconstructed as 3 mm contiguous slices from lung apex to base. Scans were photographed in windows appropriate for pulmonary parenchyma (level –600, width 1500) and mediastinum (level 40, width 400). CT showed extensive consolidation in both lungs, especially the left lower and right middle lobes (Fig. 2). The attenuation value of the consolidation ranged from –40 to –129 HU. There was a left-sided pleural effusion and extensive pleural thickening.

The diagnosis of lipid pneumonia was made on the basis of the CT finding of pulmonary consolidation of fat density in the context of previous mineral oil exposure. Apart from drainage of the pleural fluid treatment was supportive. The intercostal drain was removed after 10 days and the patient was discharged home. Follow-up at 3 months showed no reaccumulation of the effusion.

DISCUSSION

Haemorrhagic pleural effusion in association with exogenous lipid pneumonia secondary to mineral oil in adults has not, to our knowledge, been previously reported. Simple pleural effusions have been described [5]. Lipinski *et al.* [6] analysed chest radiographic findings in 11 cases of lipid pneumonia and found small pleural effusions in three. There have, however, been many series in which pleural effusions have not been documented [4,7].

Chest radiographic findings in lipid pneumonia are diverse

Author for correspondence: Dr Karen E. Paisley, Department of Clinical Radiology, Royal Devon and Exeter NHS Trust, Barrack Road, Exeter, Devon EX2 5DW, UK. Tel: +44-1752-777111; Fax: +44-1752-763277; E-mail: k.paisley@virgin.net

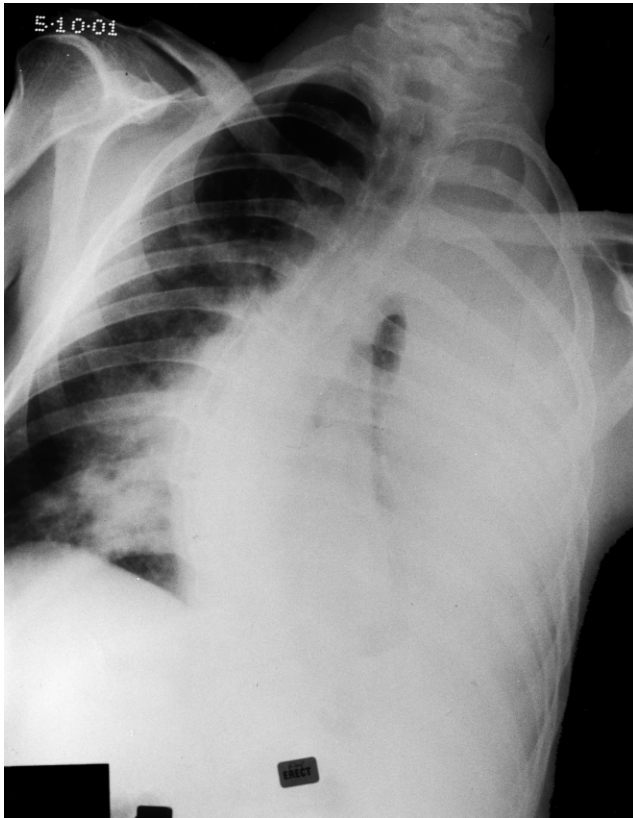
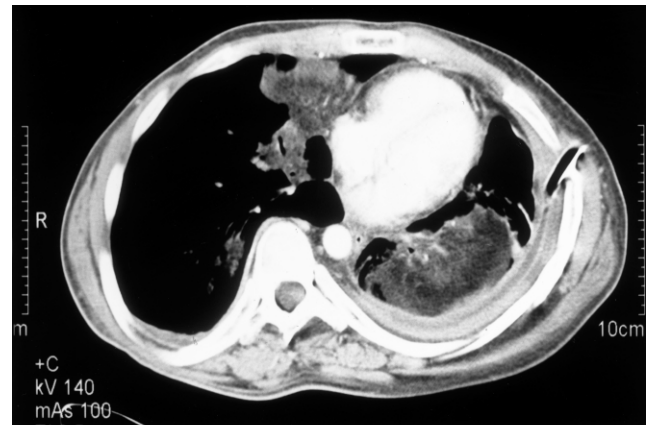


Fig. 1 – Chest radiograph on admission.

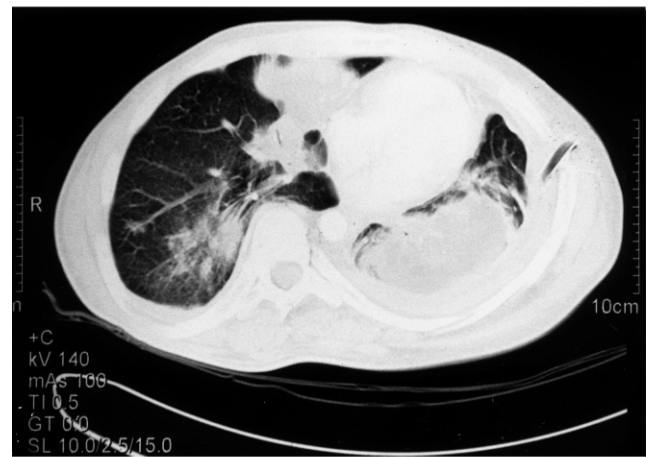
and non-specific [4,6]. Appearances range from solitary granuloma, localized or multiple areas of consolidation often with ill-defined irregular boundaries, to extensive subacute bronchopneumonia. Cavitation has been described [5]. A reticular pattern can occur as the emulsified oil leaves the alveoli and enters the interstitium and lymphatics resulting in fibrosis, volume loss and occasional lymph node involvement.

The most characteristic CT finding of lipoid pneumonia is lung consolidation of fat attenuation [7,8]. Indeed, with the appropriate exposure history, this can establish the diagnosis [3, 9]. However, this characteristic appearance is not always found. True fat density was demonstrated in only three out of seven patients in one series [2]; three of the remaining patients had densities greater than fat but less than soft tissue which is not specific and has been described in bronchoalveolar carcinoma and bacterial pneumonia. Other series have documented fat density in two out of 16 [10] and two out of six cases, respectively [7]. This discrepancy has been explained by the fact that attenuation coefficients are average values from both lipids within alveoli and surrounding inflammatory exudates.

Recently a crazy-paving pattern has been described on high resolution CT (HRCT) in patients with lipoid pneumonia [11]. This comprises patchy well-defined areas of ground glass attenuation with superimposed septal thickening. Laurent *et al.* [2] in a retrospective review of HRCT findings in seven patients with lipoid pneumonia found one or more areas of mostly central consolidation ($n = 6$), ground glass opacities ($n = 6$), thickened interlobular septa ($n = 6$), crazy-paving pattern ($n = 5$), centrilobular interstitial thickening ($n = 5$) and small



(a)



(b)

Fig. 2 – CT performed, after insertion of intercostal drain, with 3 mm collimation showing widespread consolidation of fat density in left lower and right middle lobes (a mediastinal windows, b lung windows at same level) with pleural thickening and a little remaining haemothorax.

cystic areas ($n = 1$). Lee *et al.* [3] in a retrospective review of 25 patients with lipoid pneumonia resulting from squalene aspiration found that all had abnormal parenchymal opacities on HRCT. They involved multiple lobes in 22 (especially right middle and both lower lobes) and could be classified into three types: (1) diffuse ground glass opacity ($n = 10$); (2) consolidation ($n = 7$); and (3) interstitial ($n = 5$) manifesting as irregular thickening of interstitium and parenchymal distortion. In a further series of six patients [7] CT demonstrated diffuse parenchymal consolidation in three cases, localized areas of consolidation with surrounding fibrosis in two cases, and subpleural fibrosis in one case. Interestingly mediastinal lymphadenopathy (> 1 cm short axis) was seen in two cases (right paratracheal and pretracheal node, respectively).

Magnetic resonance imaging (MRI) has been used in the diagnosis of lipoid pneumonia [12] and shows high signal on T1 with a slow decrease of signal on T2, findings characteristic of blood or fat. However MRI findings are not as specific as CT and the lower spatial resolution makes it more difficult to demonstrate thickened septa and ground glass opacities at the periphery of pulmonary consolidation areas.

In its nodular form lipoid pneumonia can mimic bronchogenic carcinoma [4] especially when classical CT findings of low attenuation are absent. Indeed the diagnosis has sometimes only been made retrospectively after thoracotomy. Although surgical resection for suspected cancer, fine-needle aspiration cytology or lung biopsy specimens have previously provided most diagnoses of lipoid pneumonia, clinical awareness, characteristic radiology and, if required, sputum or bronchial washing examination for lipid-laden macrophages can lead to the correct diagnosis without further invasive procedures.

The natural history of lipoid pneumonia is difficult to determine. While acute, occasionally fatal, cases can occur the disease is usually indolent. Concurrent debilitation or continued oil use are associated with more progressive disease. Treatment comprises cessation of exposure, treatment of complicating infection and supportive care. Some improve after steroids but in others steroids have little effect. Typically, on cessation of exposure, symptoms improve [11] but radiological features can persist [5] or deteriorate as in this case.

Reported complications include superinfection with atypical mycobacteria, aspergillus or cryptococcus, pneumothorax and hypercalcaemia [14]. Lung cancer has been described in areas of pre-existing lipoid pneumonia [13] although the association is rare. Radiographic diagnosis of the malignant transformation is difficult and consequently prognosis is poor. It is prudent to suspect malignancy when previously stable pulmonary opacities enlarge or cavitate or when new pulmonary lesions, lymph node enlargement, atelectasis or pleural effusions appear. Clearly this was a concern in our patient, particularly in view of the haemothorax, but no evidence of malignancy was found.

In conclusion, we have reported a case of lipoid pneumonia which presented with haemorrhagic pleural effusion. The CT finding of lung consolidation of fat density was sufficient to

make the diagnosis, avoiding the need for invasive diagnostic procedures.

REFERENCES

- 1 Laughlen GF. Studies on pneumonia following nasopharyngeal injections of oil. *Am J Pathol*, 1925;1:407–414.
- 2 Laurent F, Philippe JC, Vergier B, Granger-Vernon B, Darpeix B, Vergeret J, Blanc P, *et al*. Exogenous lipid pneumonia: HRCT, MR, and pathologic findings. *Eur Radiol*, 1999;9:1190–1196.
- 3 Lee JS, Im J-G, Song KS, Seo JB, Lim TH. Exogenous lipid pneumonia: high resolution CT findings. *Eur Radiol*, 1999;9:287–291.
- 4 Kennedy JD, Costello P, Balikian JP, Herman PG. Exogenous lipid pneumonia. *AJR Am J Roentgenol*, 1981;136:1145–1149.
- 5 Genereux GP. Lipids in the lungs: radiologic–pathologic correlation. *J Can Assoc Radiol*, 1970;21:2–15.
- 6 Lipinski JK, Weisbrod GL, Sanders DE. Exogenous lipid pneumonitis. *J Can Assoc Radiol*, 1980;31:92–98.
- 7 Lee KS, Muller NL, Hale V, Newell JD, Jr., Lynch DA, Im J-G. Lipoid pneumonia: CT findings. *J Comput Assist Tomogr*, 1995;19:48–51.
- 8 Joshi RR, Cholaneril JV. Computed tomography in lipoid pneumonia. *J Comput Assist Tomogr*, 1985;9:211–213.
- 9 Wheeler PS, Stitik FP, Hutchins GM, Klinefelter HF, Siegelman SS. Diagnosis of lipoid pneumonia by computed tomography. *JAMA*, 1981; 245:65–66.
- 10 Furuya MEY, Martinez I, Zuniga-Vasquez G, Hernandez-Contreras I. Lipoid pneumonia in children: clinical and imagenological manifestations. *Arch Med Res*, 2000;31:42–47.
- 11 Franquet T, Gimenez A, Bordes R, Rodriguez-Arias JM, Castella J. The crazy-paving pattern in exogenous lipid pneumonia: CT-pathologic correlation. *AJR Am J Roentgenol*, 1998;170:315–317.
- 12 Carette MF, Grivaux M, Monod B, Capeau F, Lebreton C, Bigot JM. MR findings in lipoid pneumonia. *AJR Am J Roentgenol*, 1989;153: 1097–1098.
- 13 Felson B, Ralaisomay G. Carcinoma of the lung complicating lipoid pneumonia. *AJR Am J Roentgenol*, 1983;141:901–907.
- 14 Spickard A, Hirschmann JV. Exogenous lipid pneumonia. *Arch Intern Med*, 1994;154:686–692.